Hyperkalemia

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Potassium

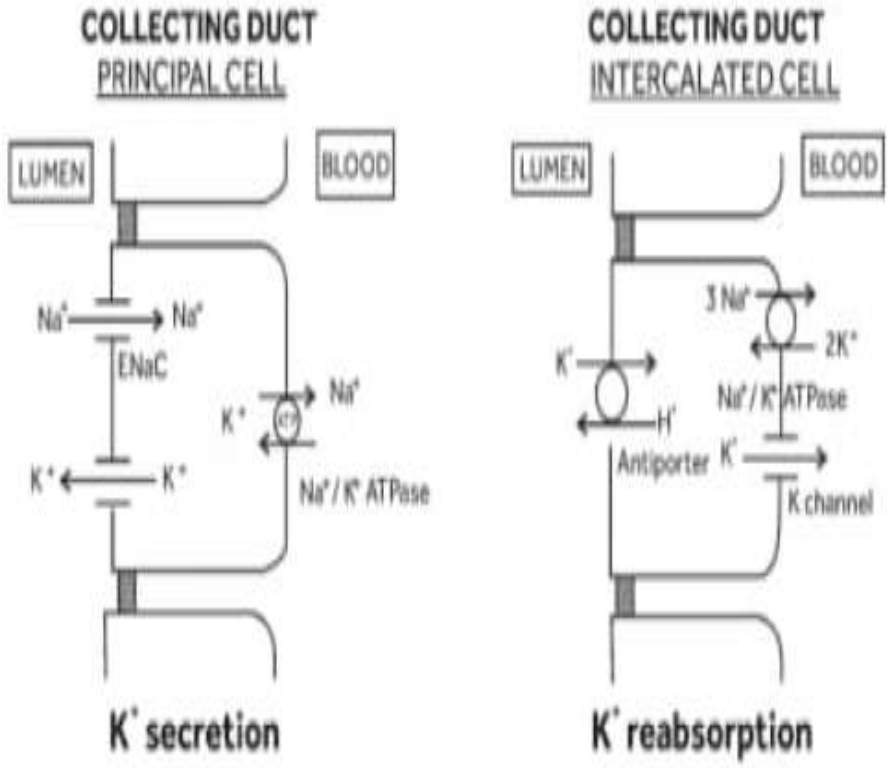
- Total body K -50mEq/Kg
- Predominantly an intracellular cation.
- 98% is in the intracellular compartment and majority in the skeletal muscle.
- The normal serum level is 3.5-5.5mEq/L. Higher levels may be seen in newborns and young infants

Role of potassium

- Essential for growth
- Maintaining the resting membrane potential of skeletal, smooth, cardiac muscle and nerves
- Helps to regulate cellular volume as well as intracellular calcium content

Regulation of Potassium levels

- 90% excreted in urine and 10% GIT, sweat
- Most of the filtered K is absorbed
- In the DCT and cortical collecting duct K is secreted into the tubular lumen in exchange with Na and H ions.
- Aldosterone acts on the collecting duct where it stimulates sodium movement from the tubules into the cell.
- This leads to a negative charge in the tubular lumen promoting potassium excretion.

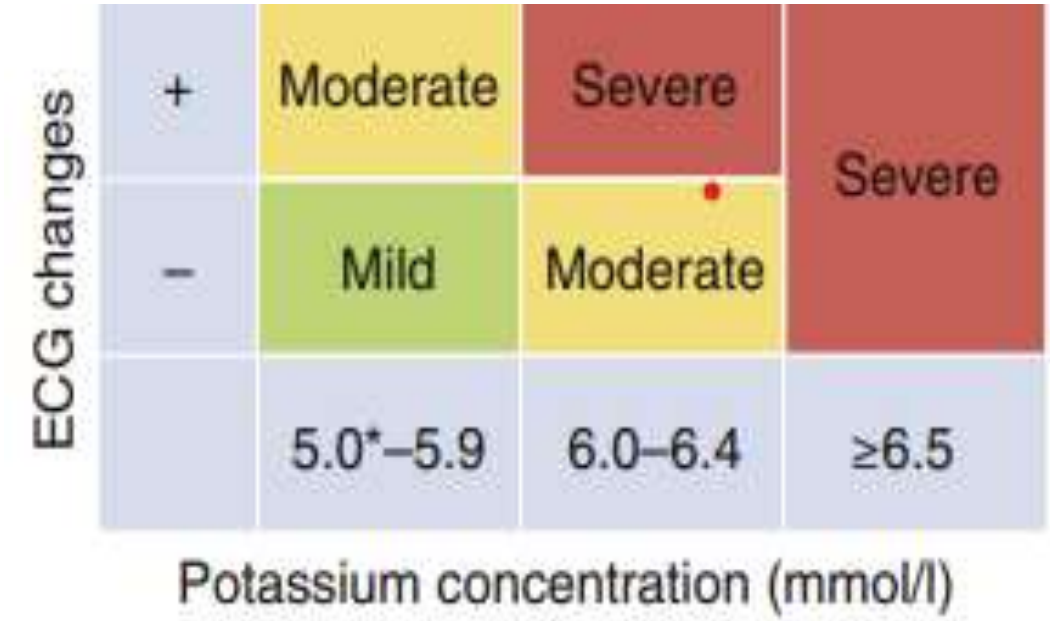


Definition

- Serum or plasma concentration of K >5.5mEq/L ; in neonates > 6mEq/L (serum K is 0.1-0.7 mmol/L higher)

- Mild hyperkalemia - 5.5-6 mEq/L.
- Moderate hyperkalemia - 6 -7 mEq/L.
- Severe hyperkalemia - >7 mEq/L

Daly, K., & Farrington, E. (2013). *Hypokalemia and Hyperkalemia in Infants and Children: Pathophysiology and Treatment. Journal of Pediatric Health Care, 27(6), 486–496.* doi:10.1016/j.pedhc.2013.08.003



Severity of acute hyperkalemia: expert opinion-
k classification. *5.0 or upper limit of normal range. ECG,
diogram.

Catherine M. Clase, Juan-Jesus Carrero , David H. Ellison4 , Morgan E. Grams et al. Potassium homeostasis and management of dyskalemia in kidney diseases: conclusions from a Kidney Disease: Improving Global Outcomes (KDIGO) Controversies Conference. Kidney International (2020) 97, 42–61

Causes of hyperkalemia

Transcellular shifts	Decreased K excretion	Increased production followed by transcellular shift*	Increased K intake
Metabolic acidosis	AKI, CRF	Tumor lysis syndrome	IV/oral
DKA	Renal tubular disease (pseudohypoaldosteronism type I and II, type 4 renal tubular acidosis,obstructive uropathy, sickle cell disease)	Excessive trauma	Blood transfusions
Lactic acidosis	Primary adrenal disease: CAH, hypoaldosteronism	Rhabdomyolysis(Crush injuries,convulsion,infection)	High dose penicillin G
Drugs-succinyl choline,beta blockers, digoxin	ACE inhibitors,ARB, K sparing diuretics,trimethoprim,NSAID	Hemolysis,GI bleed	Parenteral nutrition
Hyperosmolality(mannitol)	Hypovolemia	Malignant hyperthermia	
Hyperkalemic periodic paralysis			

Symptoms

- Asymptomatic
- Muscle weakness to ascending flaccid paralysis.
- Palpitations, syncope, arrhythmia and sudden cardiac arrest.
- Respiratory depression
- Ileus
- Paresthesia
- Jaundice

Approach- Step1

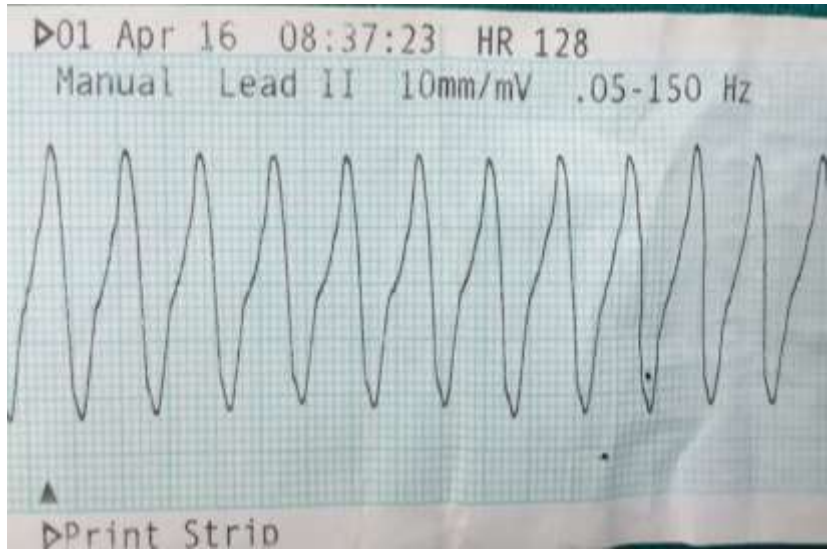
- Find out if the patient is symptomatic. Connect to cardiac monitor and take a 12 lead ECG. Look for ECG changes. If present- start treatment.

ECG changes

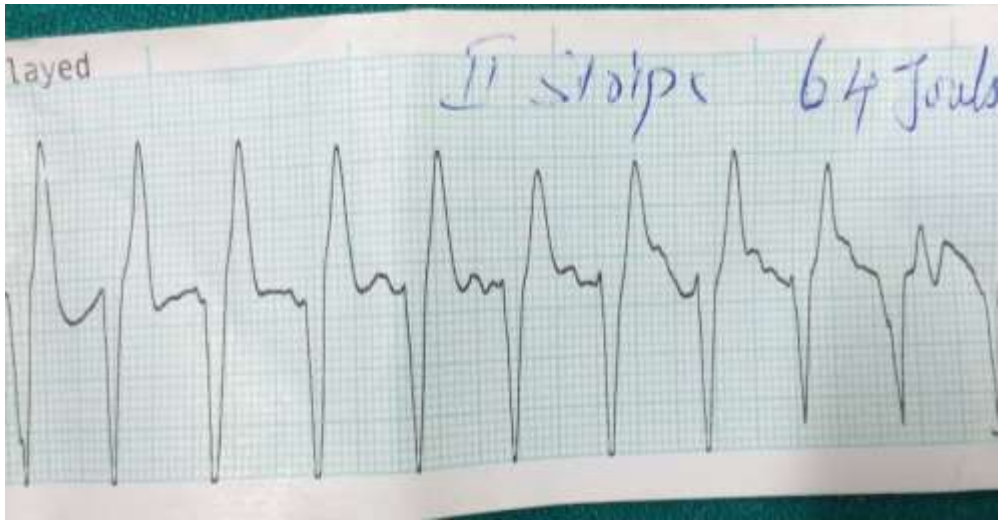
- Tall, peaked T waves
- Prolonged PR interval
- Progressive widening of QRS
- Sine wave((fusion of QRS and T wave)
- VT, VF, asystole

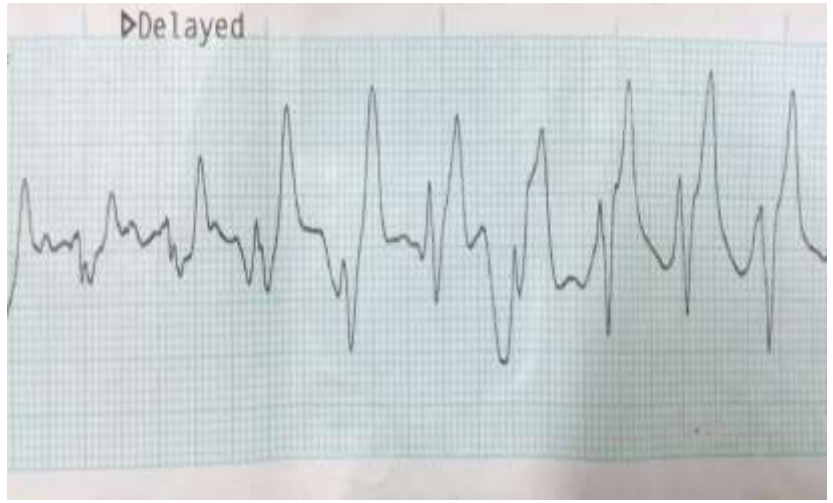
A normal ECG does not exclude risk for arrhythmia, as life threatening arrhythmia can occur without warning

ECG changes

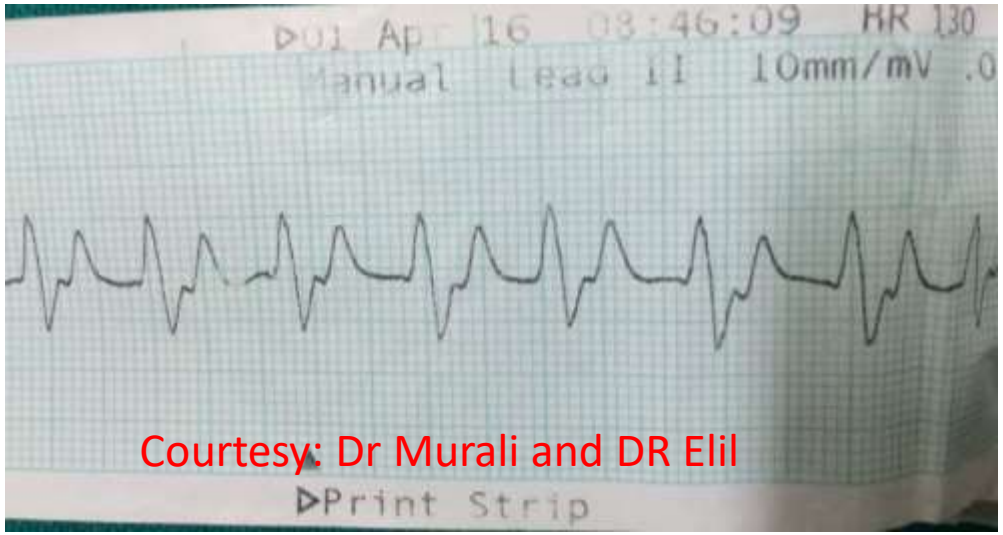


K-9.5mEq/L
Urea-156mg
Cr- 5.6mg

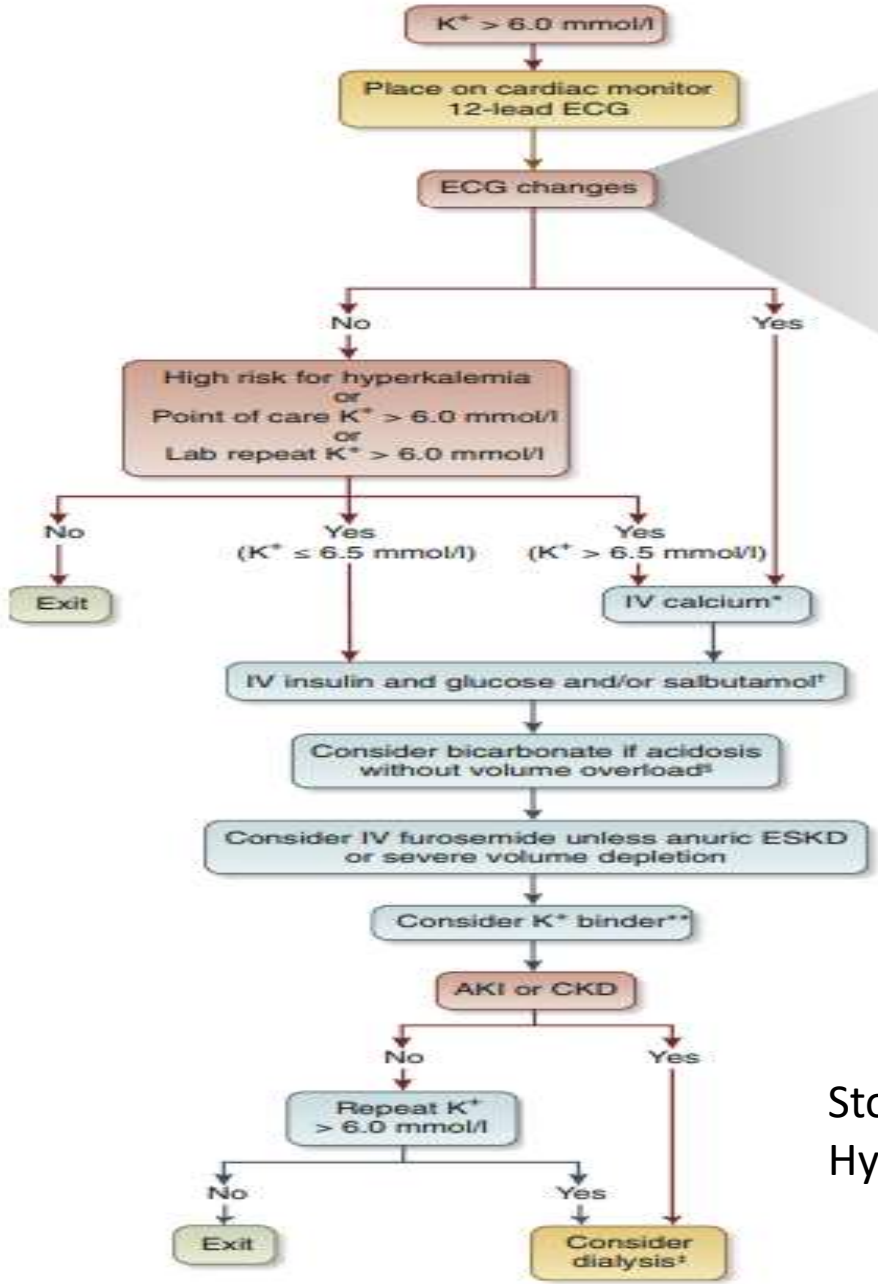




Horse-shoe kidney; bil.ureterohydronephrosis and chronic cystitis



Courtesy: Dr Murali and DR Elil



Serum potassium	Expected ECG abnormality
5.5–6.5 mmol/l	Tall, "peaked" T waves with narrow base, best seen in precordial leads
6.5–8.0 mmol/l	Peaked T waves Prolonged PR interval Decrease amplitude of P waves Widening of QRS complex
>8.0 mmol/l	Absence of T wave Intraventricular blocks, fascicular blocks, bundle branch blocks, QRS axis shift Progressive widening of QRS resulting in bizarre morphology "Sine wave" patterns (sinoventricular rhythm), VF, asystole

Airway
Breathing
Circulation

Assess and manage underlying cause

Volume
GFR
Acid-base
Monitor glucose

Stop all K containing fluids and drugs that cause Hyperkalemia. Avoid blood transfusions

Kidney International (2020) 97, 42–61

Monitoring

- Continuous cardiac monitoring and serial ECGs till K is normal
- Hourly K initially
- Blood glucose every 30 min if on insulin dextrose infusion
- I/O chart
- Electrolytes , blood gas

Step 2

- If patient is asymptomatic and does not have setting of hyperkalemia rule out pseudohyperkalemia

Pseudohyperkalemia

- Heel prick, prolonged tourniquet application, fist clenching, using a small bore needle and syringe to sample blood- hemolysis
- Restraining the limb in a crying agitated child can result in repetitive limb movement and muscle contraction
- Blood sampling proximal to an intravenous line with potassium containing fluid
- Leucocytosis and thrombocytosis

Step-3 History

- H/o burns, trauma leading to crush injury, diabetes, excess potassium intake
- Palpitations, fasciculations, syncope and parasthesias
- Drug intake by family members(accidental poisoning) ,child

History…..

- H/o renal disease- oliguria, anuria, edema, hypertension
- H/o ambiguous genitalia in a female child, recurrent shock, failure to thrive in a boy may suggest primary adrenal disease like congenital adrenal hyperplasia
- H/o blood transfusion
- History suggestive of urinary tract infection

History……

- H/o episodes of paralysis with family history (familial hyperkalemic periodic paralysis)
- H/o surgery, anesthesia (malignant hyperthermia)
- H/o chemotherapy in a child with a large tumor load

History…….

- Color of the urine(cola colored in AGN, hemoglobinuria, rhabdomyolysis)
- Bloody stools may occur in HUS
- Drug intake
- Family history of similar disorder (Single gene disorders causing hyperkalemia, malignant hyperthermia, neuromuscular disorders)

Step-4 Clinical examination

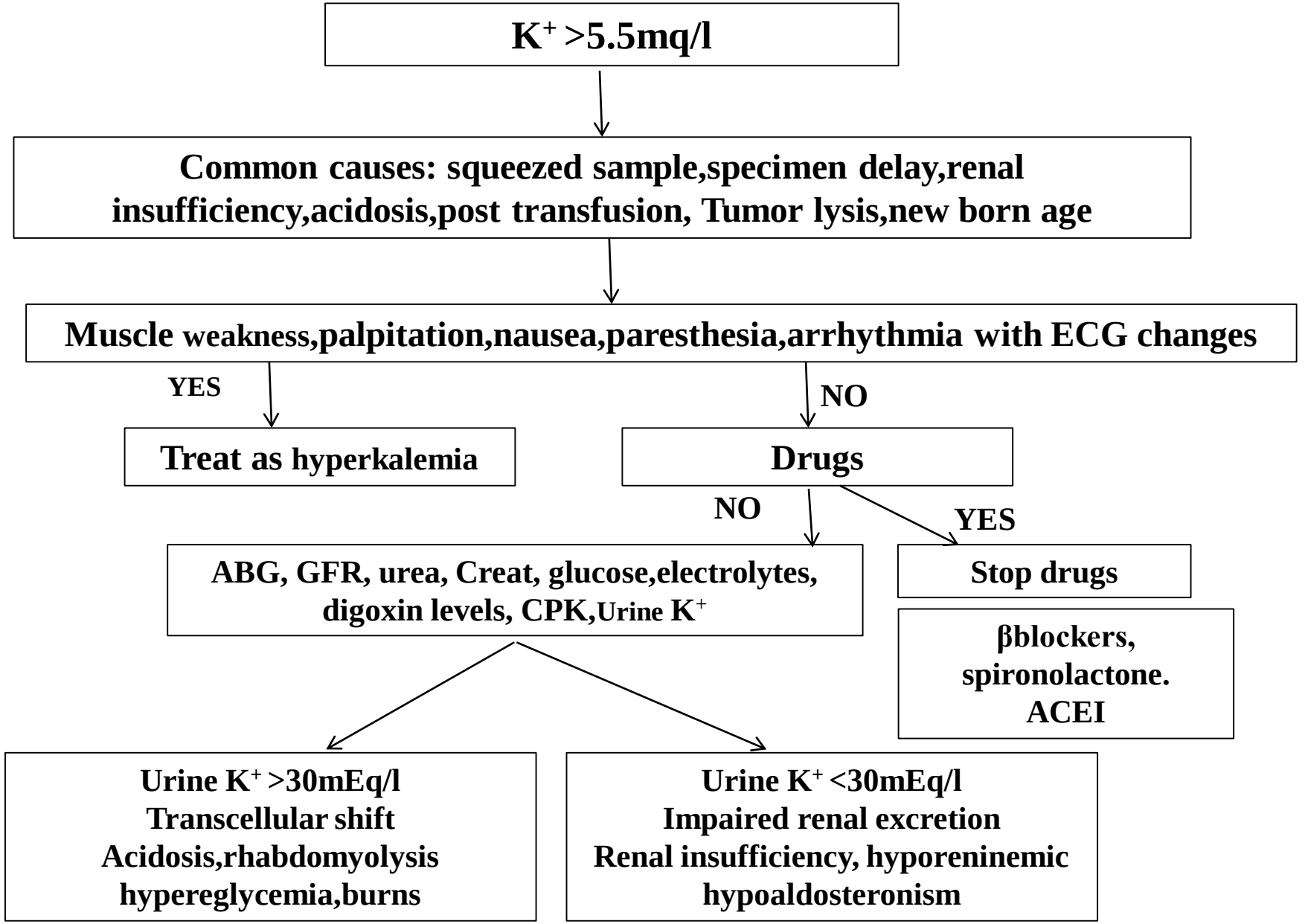
- Edema, acidotic breathing, short stature, pallor, rickets, hypertension(CKD)
- Ambiguous genitalia, hyperpigmentation (addisons disease, adrenoleukodystrophy)
- Hepatosplenomegaly, lymphadenopathy (leukemia, lymphoma)
- Muscle tenderness(rhabdomyolysis)

Step- 5 Investigations based on provisional diagnosis

- CBC
- Smear
- Urea, creatinine
- Blood glucose
- Electrolytes (Na, HCO_3, Ca, P)
- Urine analysis for proteinuria, casts
- Venous blood gas
- CPK, urine for myoglobin
- Uricacid
- USG abdomen
- urine potassium and sodium
- Endocrine workup – 17-OH progesterone, aldosterone, renin, angiotensin, cortisol, 21-hydroxylase, and 11-beta-hydroxylase

Step 6 Specialised investigations: Plasma renin activity and aldosterone

Plasma renin activity	Aldosterone concentration	Interpretation
Low	Low	Intrinsic renal disease
High	Low	CAH, hypoaldosteronism
High	High	Pseudo hypoaldosteronism


K+ >5.5mq/l
Common causes: squeezed sample,specimen delay,renal insufficiency,acidosis,post transfusion, Tumor lysis,new born age
Muscle weakness,palpitation,nausea,paresthesia,arrhythmia with ECG changes
YES
NO
Treat as hyperkalemia
Drugs
NO
YES
ABG, GFR, urea, Creat, glucose,electrolytes, digoxin levels, CPK,Urine K+
Stop drugs
βblockers, spironolactone. ACEI
Urine K+ >30mEq/l
Transcellular shift
Acidosis,rhabdomyolysis
hypereglycemia,burns
Urine K+ <30mEq/l
Impaired renal excretion
Renal insufficiency, hyporeninemic hypoaldosteronism

Treatment of reversible causes of hyperkalemia

- Hypovolemia
- Salt wasting congenital adrenal hyperplasia, adrenal insufficiency (Inj hydrocortisone 100mg/sqm stat)
- Medications which impair renal excretion of K
- Potassium containing fluids or medications
- Metabolic acidosis

Management of hyperkalemia in children, UpToDate 2020

Mild hyperkalemia without ECG changes

- Stop K supplements
- Salbutamol nebulisation
- Kayexelate
- Diuretics

Chronic hyperkalemia in CKD children

- A low potassium diet
- Loop diuretic therapy
- Correction of metabolic acidosis with carbonate therapy
- Avoidance of drugs that increase potassium levels(Spironolactone, ACE inhibitors or ARBs.
- RRT

Management of hyperkalemia in children, Uptodate 2020

Thankyou